Insomnia and Hypertension

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We would like to respond to the 2 letters to the editor,[1,2] which were published in *SLEEP*, August 2009, in regard to our study**, "**Insomnia with objective short sleep duration is associated with a high risk for hypertension."[3]

The objective of our study was to assess the cross-sectional association of two prevalent conditions, i.e., insomnia and hypertension, in a general-population sample. Our study demonstrated a strong and significant association between insomnia with objective short sleep and hypertension. Given the high prevalence of hypertension in the general population, we did not intend to imply that the odds ratio is a reliable estimate of the relative risk for this condition in insomniacs with short sleep, versus the reference group. In less technical terms, our odds ratios indicate that the odds or the likelihood of having hypertension is 5-fold higher in the insomnia–short sleep-duration group versus the control group. Thus, we agree with Sainani et al., that the phrase "a risk for hypertension 500% or 350% higher," although commonly used, is grammatically incorrect and may lead to misinterpretation.

Dr. Krakow suggests that, in our study, we underestimated the severity of obstructive sleep apnea because we used only thermocouple technology. In our study, in contrast with many previous reports, subjects were evaluated for sleep apnea, the prevalence of which was the primary goal of our project, with technology very similar to other large epidemiology cohorts, i.e., Sleep Heart Health Study and Wisconsin cohort. In our analysis, for the association of insomnia with hypertension, we controlled even for mild forms of apnea, i.e., an apnea-hypopnea index of 5 or less, which usually are not treated in most clinical settings. Dr. Krakow also suggests that we did not assess for other forms of the apnea spectrum disorder, such as upper airway resistance syndrome. This syndrome is usually associated with sleep fragmentation but not with apneas or hypopneas based on standard definition. In our study, we controlled for indices of sleep fragmentation, i.e., number of awakenings, number of sleep-stage changes, and percentage of stage 1 sleep, and the results remained significant and strong (see results section, fourth paragraph). Further, the author states that upper airway resistance syndrome occurs more frequently in subjects with short sleep duration. We do not know of any evidence supporting such a claim. However, even if we accept such a claim, how can we explain that the adjusted odds ratio for subjects with short sleep duration, i.e., less than 5 hours, is lowest in those with no sleep complaint (1.13, 95% confidence interval 0.79-1.62), intermediate in those with "poor sleep" (a milder form of insomnia) (2.43, 95% confidence interval 1.36-4.35), and highest in those with chronic insomnia (5.12, 95% confidence interval 2.22-11.79)? This strong dose-response association between the joint effect of insomnia with short sleep duration and hypertension can only be explained by the difference in the severity or chronicity of the insomnia complaint and not by the "underestimation of the severity of sleep apnea" and the lack of detection of upper airway resistance syndrome that should be equally distributed, based on the author's argument, in these 3 groups. Notably, our new study on the same population showed a similar pattern on the association between insomnia–short sleep duration and diabetes.[4]

The difficulties in treating mild or moderate sleep apnea (apnea-hypopnea index < 30) are well recognized, whereas the effects of current standard treatment, i.e., continuous positive airway pressure, on hypertension in this population are very doubtful, albeit very costly.[5,6] Effective treatment for a large group of insomniacs with short sleep duration may be crucial to protect them from the significant cardiometabolic problems associated with this very prevalent disorder.

DISCLOSURE STATEMENT

The Authors have indicated no financial conflicts of interest.

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